Bandolier 89

What do we think?
What do we know?
What can we prove?

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Flat tyres - only at the bottom

"*Croire que l'on sait est souvent pis que savoir qu'on ignore*" - believing that one knows is often worse than knowing that one is ignorant. ***Bandolier*** saw this concerning the date of composition of the Chanson de Roland, a famous medieval ballad (Medieval Life, Spring, 2001). The trouble is knowing what we don't know.

This month's ***Bandolier*** tries to help. Prognosis is a difficult area to find good quality knowledge. Methods can be used that are similar to diagnostic testing, where it's all about finding criteria positively associated with outcome, combining them in a decision rule, and testing that rule in another population. That has been done for the risk of stroke with AF, and one year mortality for older people in hospital. Both use patient characteristics, and are helpful because an estimate of risk makes planning treatment that bit easier.

Then there are two treatment areas folk have asked about - ACE inhibitors in renal disease and diabetes, and breast reduction surgery. There were systematic reviews, or large randomised trials, or both. There were fascinating methodological issues, but more fascinating was just how fast one can go from ignorance to some semblance of understanding.

Finally, there's the appliance of anti-bias detection. That helps protect against other people's ignorance. We give a worked example of acupuncture for stroke. There's a space limit in print, so it is supported by an article on the Internet site in HTML and downloadable PDF formats.

Electronic Bandolier

The electrons have been whizzing again, with 90,000 visitors a week. On the ***Bandolier*** Internet site this month several new features:

We have pulled together information on **atrial fibrillation** to create a new sub site. It has information on prevalence, prevention, treatment and management issues. Over the rest of the year we will add more material.

In **sexual health** we have reviewed currently available treatments for erectile dysfunction, with a league table of relative efficacy. The trials occasionally have interesting twists, and we try and explain what we think they are.

In **healthy living** you will find a section on how to lose weight. We've had some nice comments on this, and will have it as a downloadable PDF when time permits.

ACE-INHIBITORS FOR RENAL DISEASE AND DIABETES?

Bandolier has heard it said that most patients with chronic renal disease or diabetes should be taking ACE inhibitors. Evidence seems to be accumulating, in the shape of systematic reviews and large randomised trials. A brief review, then, of some of the evidence on outcomes, seems in order.

Chronic renal disease [1]

This review used a MEDLINE search (to June 1999) for studies of ACE inhibitors in chronic renal disease, supplemented with hand searching of abstracts, examination of reference lists of reviews, and solicitation of pharmaceutical companies. A broad definition of chronic renal failure was used, consisting of albuminuria greater than 30 mg/day (21 µg/minute) and/or elevated serum creatinine (more than 106 µmol/L for women and 124 µmol/L for men).

Trials were eligible if they were randomised, had a parallel comparison of ACE inhibitor with placebo, had a minimum follow-up of one year and studied adults. Information unavailable in the published reports was requested from the original authors.

Results

There were nine studies with 650 patients with chronic renal failure and microalbuminuria initially, and who were treated with ACE inhibitor for one to five years (average three years). ACE inhibitors were enalapril 10 or 20 mg, captopril 100 mg and lisinopril 10 mg. The outcome was development of macroalbuminuria. The risk of developing macroalbuminuria was lower with ACE inhibitor (10%) than with placebo (24%) (Figure 1; Table 1). Treating seven patients with chronic renal failure and microalbuminuria for three years with an ACE inhibitor would prevent one of them developing macroalbuminuria.

In this issue

Table 1: Chronic renal disease

Patients at onset	Outcome	Number of trials	Number/total (%) ACE inhibitor	Number/total (%) Placebo	Relative risk (95% CI)	NNT (95% CI)
Microalbuminuria	Developed macroalbuminuria over 3 years	9	30/326 (10)	74/316 (24)	0.4 (0.3 to 0.6)	7 (5 to 12)
Overt proteinuria	End-stage renal disease or doubled creatinine over 2 years	7	115/700 (17)	194/689 (28)	0.6 (0.5 to 0.7)	9 (6 to 14)

Figure1: Developing macroalbuminuria

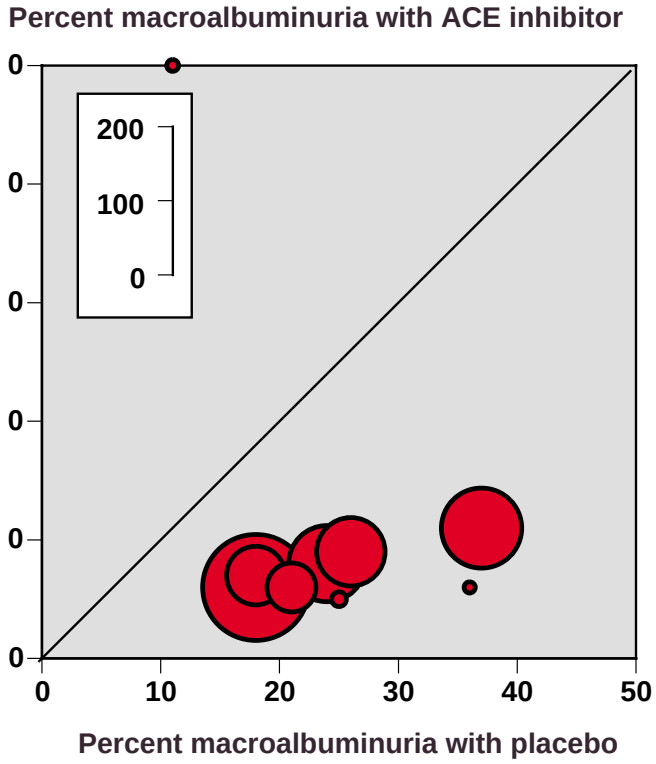
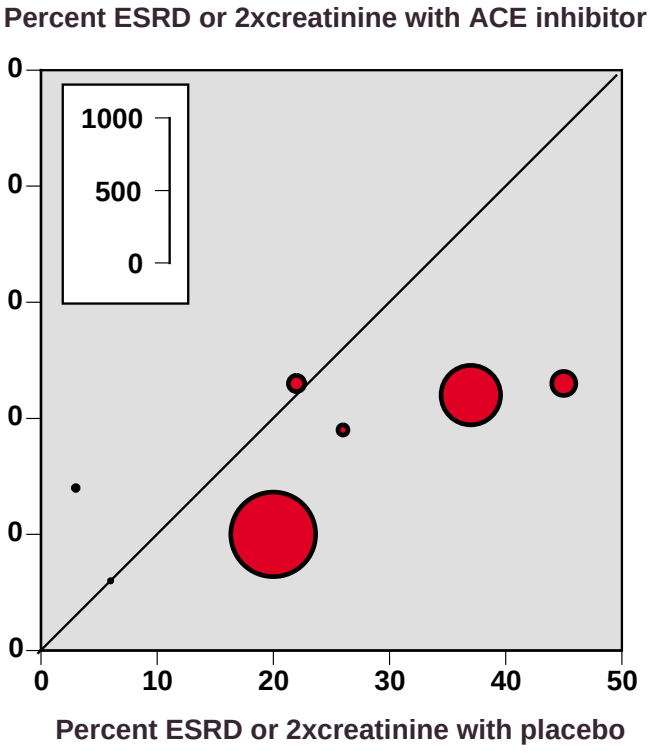


Figure 2: Developing end-stage renal disease



There were seven studies with 1,400 patients with chronic renal failure and overt proteinuria initially, and with ACE inhibitor for one to three years (average two years). ACE inhibitors were enalapril 5-40 mg, captopril 75 mg and ramipril 2.5-5 mg and benazepril 10 mg. The outcome was development of end stage renal disease or doubling of serum creatinine. The risk of developing this outcome was lower with ACE inhibitor (17%) than with placebo (28%) (Figure 2; Table 1). Treating nine patients with chronic renal failure and overt proteinuria for two years with an ACE inhibitor would prevent one of them developing end stage renal disease or doubling their serum creatinine.

Diabetes [3]

The Heart Outcomes Prevention Study (HOPE [2]) was a large (9,000 patient) study examining whether the addition of an ACE inhibitor to current medical regimen of patients with vascular disease or diabetes can lower the risk of cardiovascular events. It showed that 10 mg daily ramipril in high risk patients over five years reduced the risk of myocardial infarction, stroke, or cardiovascular death. Treating 1000 patients with ramipril for four years prevented about 150 events in about 70 patients. A separate analysis showed the effects in the 40% of patients with diabetes [3].

Patients recruited were 55 years or older, who had a history of cardiovascular disease, diabetes, plus at least one other cardiovascular risk factor (total cholesterol above 5.2 mmol/L, HDL cholesterol ≤0.9 mmol/L, hypertension, microalbuminuria, or current smoking). They were randomised to 10 mg ramipril or matched placebo, and were followed for 4.5 years. The main outcome was myocardial infarction, stroke, or cardiovascular death, a combined outcome of bad things. These were also examined individually, with a number of secondary outcomes.

Results

The main results are shown in Table 2, as percentages of events occurring. Those shown were statistically significant. When major cardiovascular and microvascular events were taken into account, 15 high-risk people with diabetes would have to be treated with ramipril for 4.5 years to prevent one of them having a myocardial infarction, stroke, cardiovascular death, admission to hospital for heart failure, a revascularisation procedure, development of overt nephropathy, laser therapy for retinopathy, or renal dialysis.

Table 2: ACE inhibitors for diabetes

Outcome	Percent occurring	
	Ramipril	Placebo
Number of patients	**1808**	**1769**
Combined primary outcome	15.3	19.8
MI	10.2	12.9
Stroke	4.2	6.1
Cardiovascular death	6.2	9.7
Total mortality	10.8	14
Revascularisation	14	16.4
Overt nephropathy	6.5	8.4
Any heart failure	11	13.3
TIA	4.4	5.9

Type 1 diabetes [4]

Should all patients with type 1 diabetes mellitus and microalbuminuria receive ACE inhibitors? This question was answered by a meta-analysis [4] performed on individual patient data. A MEDLINE search supplemented by reference lists and other meta-analyses identified 12 studies meeting inclusion criteria. These were normotensive diabetics with microalbuminuria (20 to 200 µg/minute) treated with ACE inhibitor or placebo in a randomised trials with at least one year follow up. Information about individual patients was requested from original investigators. The primary outcome was the change in albumin excretion rate.

Results

There were 12 trials with 700 patients. Early analysis showed a time-dependent response, and so the analysis was restricted to a two-year follow up from 10 of the 12 studies (646 patients). There was a marked beneficial effect of ACE inhibitors, with albumin excretion rate 50% lower at two years in treated versus untreated patients. The effect was highly dependent on baseline albumin excretion rate, with a small effect (18%) at the lower boundary of the target range (20-200 µg/min) and a higher (74%) reduction at the higher boundary.

Progression to macroalbuminuria was reduced. The odds ratio was 0.4 (0.3 to 0.6). Regression to normal albumin excretion was increased. The odds ratio was 3.1 (2.2 to 4.4).

Comment

These three papers show useful clinical effects of ACE inhibitors on renal and cardiovascular outcomes in chronic renal disease and diabetes. This occurs with fixed dosing, rather than with dosing adjusted to provide the maximum blood pressure drop. Though there was some statistically lower blood pressure, it was usually less than 2 mmHg, beyond the ability of most to measure. We probably have to think beyond blood pressure for these effects. The outcomes are important. Preventing bad things happening to high risk renal or diabetic patients is good, and the ACE inhibitors appear to be effective in that.

Interesting were the different ways in which evidence was obtained. We have the contrast between meta-analysis of studies [1], of individual patients from studies forming a subgroup not defined in the original studies themselves [4], and a large subgroup within a larger overall trial [3].

All the papers are naturally full of detailed statistics. That is as it should be. Two also give results in terms our tired brains can handle. Notable is the choice of presenting information in terms of "all bad things" [3], so we know that whatever bad thing it is, treating 15 diabetic high-risk patients with ramipril for 4.5 years will prevent one of them [3]. Another [1] gives us raw data with the statistics, so we can look for ourselves and do what we want, which is how ***Bandolier*** could calculate an NNT.

But the third [4] gives us only odds ratios. It is terrific that ACE inhibitor treatment of type 1 diabetics with microalbuminuria reduces progression of macroalbuminuria and promotes return of normal albumin excretion. What is missing is any suggestion of the therapeutic effort needed to achieve this. Odds ratios just don't do this. Table 3 calculates the odds ratios for a theoretical reduction in progression to macroalbuminuria. Given 700 patients split equally between treatment and placebo, the same odds ratio (0.4) as found in the meta-analysis could be an NNT of 4 or 23. The proportion of patients benefiting could be 25% or 4%. So a plea to the academics. Good statistical methods are vital. Getting the statistical tick is the priority. But when you've got it, please express the result in ways that ordinary mortals can understand and use for their patients. If you can't do that, what's the point?

Adverse events don't receive the detailed treatment they deserve in any of these papers.

References:

1 AV Kshirsagar et al. Effect of ACE inhibitors in diabetic and nondiabetic chronic renal disease: a systematic overview of randomized placebo-controlled trials. American Journal of Kidney Diseases 2000 35: 695-707.

2 HOPE Study Investigators. Effects of an angiotensin-converting enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. New England Journal of Medicine 2000 342: 145-153.

3 HOPE Study Investigators. Effects of ramipril on cardiovascular and microvascular outcomes in people with diabetes mellitus: result of HOPE study and MICRO-HOPE substudy. Lancet 2000 355: 253-259.

4 ACE Inhibitors in Diabetic Nephropathy Trialist Group. Should all patients with type 1 diabetes mellitus and microalbuminuria receive angiotensin-converting enzyme inhibitors? Annals of Internal Medicine 2001 134: 370-379.

Table 3: Changing NNT at fixed odds ratio

Treatment (n=350)		Placebo (n=350)			
Number	%	Number	%	Odds ratio	NNT
10	3	25	7	0.41	23
20	6	50	14	0.39	12
40	11	90	26	0.39	7
60	17	120	34	0.41	6
100	29	180	51	0.39	4
150	43	230	66	0.40	4

Risk of stroke with AF

Bandolier was once made aware of the GP's crie de cour: don't tell us what treatment to use, but tell us what patients to treat! The trouble is that there's not an awful lot of good evidence around. Some rejoicing, then, when a paper helps by giving a simple clinical way of estimating the risk of stroke in patients with non-rheumatic atrial fibrillation [1].

Study

The patients in this study were 1733 people aged 65 to 95 years discharged from hospital with a diagnosis of non-rheumatic atrial fibrillation, without any treatment apart from aspirin in some. A national registry was generated using anonymous information from five quality improvement or peer review organisations serving seven US states. Medicare records could be reviewed for proper assessment and diagnosis of atrial fibrillation, documented risk factors, any therapy or comorbid conditions. Standardised abstraction forms were used, with excellent agreement between abstractors.

The study outcome was hospital admission for first ischaemic stroke, as determined by Medicare claims. The minimum follow up was 365 days, and the maximum was 1000 days.

Risk classification scheme

Two risk classification schemes used in clinical trials to classify patients with atrial fibrillation at low, medium and high risk of stroke were combined. The new scheme involved independent risk factors identified in the original two schemes: one point was given for the presence of congestive heart failure, hypertension (systolic >160 mmHg), age greater than 75 years, and diabetes, and two points given for prior cerebral ischaemia (Table 1). The name of the new classification scheme, CHAD2, is also an acronym.

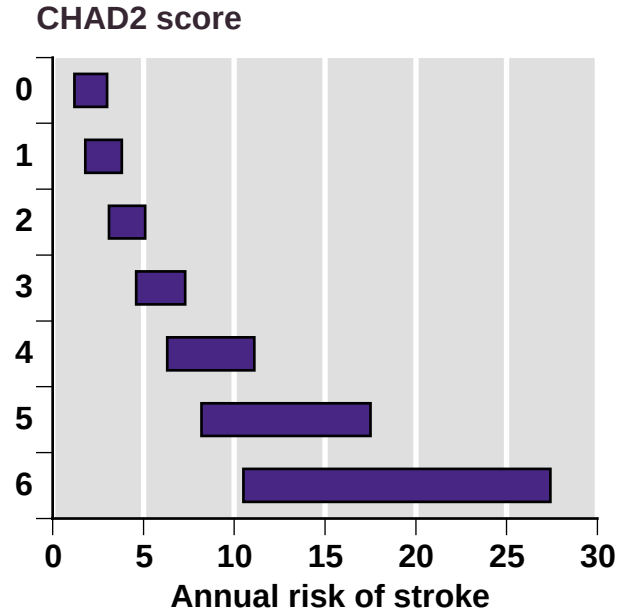
Table 1: Components of CHAD2

CHAD2 item	Points
Congestive heart failure	1
Hypertension (systolic >160 mmHg)	1
Age greater than 75 years	1
Diabetes	1
Prior cerebral ischaemia	2

Table 2: CHAD score and risk of stroke

CHAD2 score	Number of: Patients	Strokes	Adjusted annual stroke rate (95%CI)
0	120	2	1.9 (1.2 to 3.0)
1	463	17	2.8 (2.0 to 3.8)
2	523	23	4.0 (3.1 to 5.1)
3	337	25	5.9 (4.6 to 7.3)
4	220	19	8.5 (6.3 to 11.1)
5	65	6	12.5 (8.2 to 17.5)
6	5	2	18.2 (10.5 to 27.4)

Figure 1: CHAD score and risk of stroke



Results

There were 94 strokes in the 1733 patients over an average 1.2 year follow up, a crude average rate of 4.5% a year. Within the cohort, the crude rate for patients with no risk factors (120 people) was 1.2%. With increasing risk scores, the crude annual risk rose also. Figure 1 shows the 95% confidence interval of the rate after smoothing through an exponential survival model. Table 2 gives the numbers..

Comment

This is another example of a clinical scoring system being tested and showing how useful they can be. Here the very system, being an acronym, is helpful in bringing to mind important risk factors for stroke with non-rheumatic atrial fibrillation. The annual risk of stroke rose from under 2% a year with no risk factors to over 10% a year for five or six. At some point the balance tips to the use of anticoagulants.

It would be terrific to see this scoring system tested on a separate data set, but for now we have a useful way of targeting warfarin treatment at patients who will benefit most, those with a high baseline risk. Patients with a lower risk can be offered aspirin.

References:

1 BF Gage et al. Validation of clinical classification schemes for predicting stroke. Results from the national registry of atrial fibrillation. JAMA 2001 285: 2864-2870.

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PROGNOSTIC INDEX FOR MORTALITY OF OLDER PEOPLE AFTER HOSPITAL ADMISSION

Older people are more likely to be admitted to hospital, and for many of them who may not previously suffered a major illness this can be a major event that defines the future course of their life. Knowing their expected mortality in the year after discharge may be helpful in planning ongoing care with them and their families, and with the primary care physicians. A new study [1] gives a simple bedside method for doing this, and also forms an exemplar of how diagnostic or prognostic tests can be developed.

Study

Patients aged 70 years and older admitted for two days or more to general medical services of two hospitals in Ohio, and taking part in a randomised trial, were the subjects of the study. Excluded were elective admissions, patients admitted to intensive care units, or admissions to subspecialty services. About 4% of enrolled patients had missing data, and were excluded. About a quarter of all admissions between 1993 and 1997 were used.

Patients from one hospital were used as a derivation cohort, from which risk categories and scoring system were devised. Patients from the other hospital were used as a validation cohort to test the rules.

Predictors

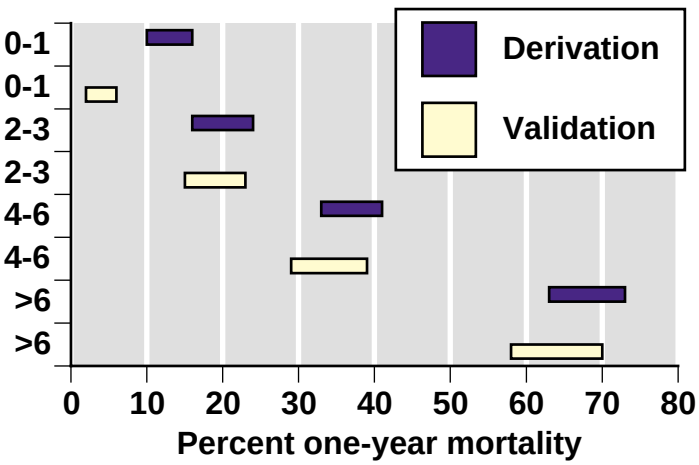
Predictors of mortality were obtained from standardised interviews with patients, or from surrogates if patients were too ill. As well as demographic information, independence in five activities of daily living was assessed. These were bathing, dressing, using the toilet, transferring from bed to chair, and eating. A person who required assistance to perform one of these activities was classified as dependent for that activity. As well as the interview, comorbid conditions and admission laboratory values were obtained from medical records by trained medical abstractors.

Table 1: Independent risk factors

Risk factor	Odds ratio (95%CI)	Points
Male sex	1.4 (1.1 to 1.8)	1
Dependent in 1-4 activities of daily living	2.1 (1.6 to 2.8)	2
Dependent in all 5 activities of daily living	5.7 (4.2 to 7.7)	5
Congestive heart failure	2.0 (1.5 to 2.5)	2
Solitary cancer	2.6 (1.7 to 3.9)	3
Metastatic cancer	13.4 (6.2 to 29)	8
Admission creatinine >265 µmol/L	1.7 (1.2 to 2.5)	2
Admission albumin 30-34 g/L	1.7 (1.2 to 2.3)	1
Admission albumin less than 30 g/L	2.1 (1.4 to 3.0)	2

The adjusted odds ratio was from multivariate logistic regression using the derivation cohort

Figure 1: Risk and scores for derivation and validation cohorts



Mortality

The outcome was death within one year of discharge from hospital. This information was obtained from follow up interviews with patients or families, or from a death index. This information was available for all patients.

Method

The relationship between the one-year mortality and each of many variables was found by logistic regression analysis. Those factors that were independently significantly associated with mortality were given points.

Results

Six risk factors were independently associated with mortality - male sex, dependency for activities of daily living, congestive heart failure, cancer, and admission creatinine and albumin values. The risk factors, the strengths of association, and points given for each factor are shown in Table 1. In the derivation and the validation cohort, increased scores were associated with an increased risk of death in the year after hospital discharge.

Comment

This is an ideal paper to read for detailed methods about developing clinical scoring systems. It has the same sort of strength as found in the development of the Ottawa ankle and knee rules (***Bandolier*** 21 and 49). It is interesting that a number of these clinical scoring systems are now appearing, predominantly from north America.

Knowing that the risk of dying in the next year is low or high may be useful to professionals planning care, or lay carers or patients. Individual cases will differ. Perhaps the real strength of this type of evidence, and scoring systems in general, is they remind us about what is important and become an ongoing quality check.

References:

1 LC Walter et al. Development and validation of a prognostic index for 1-year mortality in older adults after hospitalization. JAMA 2001 285: 2987-2994.

Breast reduction surgery - does it help?

Breast hypertrophy can be a real problem for some women. Reduction mammoplasty using various surgical techniques has become a more common treatment. Over the last 30 years about 32,000 such operations have been performed in Sweden, an average rate of about 25 per 100,000 women per year. That's about 12 a year in the average PCG, so a GP might see a case once every five years or so.

A new systematic review [1] pulls together the best available information. It informs about before and after symptoms, and is fascinating because this is one of those topics where randomised trials are rare as hen's teeth. So the question arises about dealing with different study architectures.

Systematic review

The authors [1] searched a variety of electronic databases for studies, using a number of different languages, and supplemented this with bibliographies and reviews. Studies that were case reports, abstracts, without outcomes of interest, with mixed procedures, in the setting of breast cancer, or with mixed genders, were not used. Those included had a minimum of 10 patients, were controlled trials, or case series, or historical or cross-sectional surveys, had a diagnosis of unilateral or bilateral breast hypertrophy or macromastia, used reduction mammoplasty and had clinical outcomes of interest.

The outcomes of interest were preoperative and postoperative reports of signs and symptoms and quality of life.

Results

Twenty-nine reports were included, with 4,200 women. Eighteen were observational studies, predominantly cross-sectional, and eleven were experimental studies, predominantly uncontrolled case series. The average age of women in the studies was about 36 years, with a wide range from 11 to 86 years. The average amount of breast tissue removed was about 1,500 grams from both breasts, with a range of 100 grams to 8,000 grams. Reduction was bilateral in over 90% of women. Observational and experimental studies had similar patient characteristics.

The frequency of reported symptoms before breast reduction was high (Table 1; Figure 1). Shoulder pain, and shoulder grooving caused by brassiere straps occurred in about 85% of women preoperatively, though back, neck and breast pain also occurred in over half of women before breast reduction, and intertrigo was also common. After the breast reduction the frequency of the symptoms reduced dramatically, and to about 10% on average (Figure 1; Table 1).

Comment

Breast hypertrophy is not a common condition. Most of us would rarely if ever think about it, and even less consider the problems for the women concerned. The occurrence of such a high frequency of pain in the shoulders, neck, back and breast is probably a surprise. The effect of breast reduction in reducing the frequency of these complaints is obvious, and for some symptoms, like neck and back pain, this may represent the rate in the general population.

The problem is that the quality of the studies is less than we come to expect from randomised double-blind trials. These are perhaps not appropriate in this situation, and we don't have them. What we do have is the best information available to us now, rather than the best information that might ever be available. Cries for more research are not much help when a woman walks in asking for help.

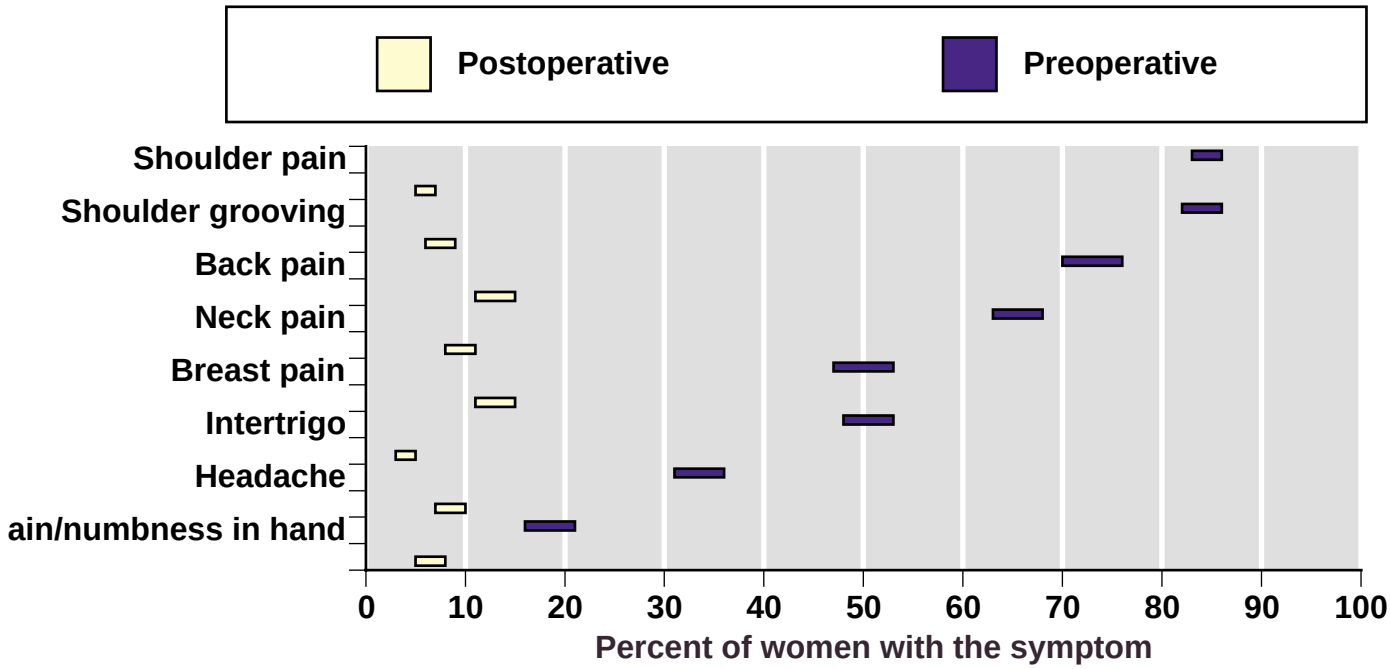
What we have is a number of studies, with information on over 4,000 women. They tell us of a big reduction in symptoms associated with breast reduction, and which makes sense from the mechanics of reduced strain on shoulders, neck and back.

We might also search for any other evidence that could help us decide whether breast reduction, on the whole, is a good thing. Studies documenting lower breast cancer risks after breast reduction [2,3] help the thinking.

Table 1: Symptoms reported before and after breast reduction surgery

	Number of:		% of women with the problem	
	Studies	Women	Preoperative 95% CI	Postoperative 95% CI
Shoulder pain	7	1829	85 (83-86)	6 (5-7)
Shoulder grooving	12	1838	84 (82-86)	8 (6-9)
Back pain	11	1153	73 (70-76)	13 (11-15)
Neck pain	11	1582	65 (63-68)	10 (8-11)
Breast pain	8	1364	50 (47-53)	13 (11-15)
Intertrigo	10	1513	50 (48-52)	4 (3-5)
Headache	7	1427	34 (31-36)	9 (7-10)
Pain/numbness in hand	4	934	19 (16-21)	7 (5-8)

Figure 1: Symptoms reported before and after breast reduction surgery



Breast reduction and cancer

Both the studies were conducted in Sweden where a first class nationwide record linking system. Cancer, death, emigration and inpatient registers could be used to identify the 32,000 women who had breast reduction between 1965 and 1999, and to determine whether they subsequently developed breast cancer. One hundred and sixty one women did develop breast cancer, with 240,000 women-years, an average 7.5 year follow up.

Results

The first analysis [2] showed that there was a 28% reduction in the risk of breast cancer in women undergoing breast reduction.

The second study [3] retrieved hospital records of 137 of the women undergoing breast reduction and 422 of 483 matched controls in the breast reduction patients with similar risk factors for breast cancer, but who had not developed breast cancer. There was an impressive inverse correlation between amount of breast tissue removed and risk of breast cancer.

Compared with women who had less than 800 grams total tissue removed, those with more than 1600 grams removed had a 76% reduction in risk (Figure 2). Risk was significantly reduced for all amounts of tissue removed over 800 grams compared with less than 800 grams. This relationship was found to be the same for age, duration of follow up, weight, and parity.

Comment

That breast reduction reduces the risk of breast cancer comes from other studies as well, though the Swedish studies are notable for their completeness and strength. There is no grand unifying theory as yet, but having less breast tissue is clearly a candidate, despite there being no evidence that breast size is independently related to the risk of breast cancer.

It is always interesting when different bits of information come together to inform on a subject. Breast reduction is one of those topics of which many of us will be only dimly aware. Then along come a handful of papers full of evidence, and we feel we know much more than most.

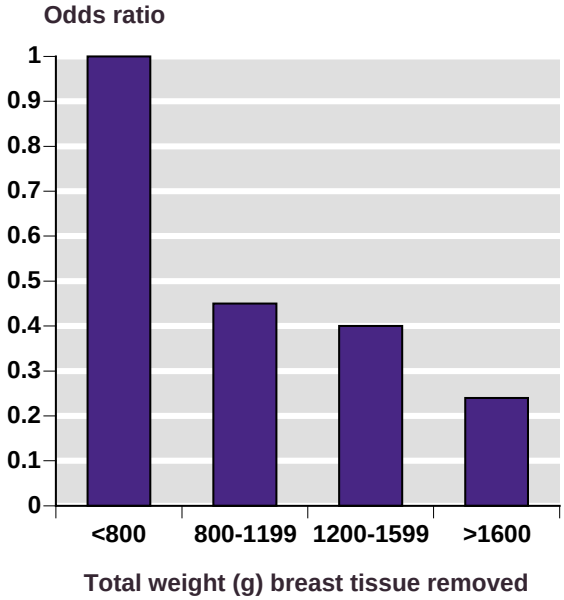
References:

1 EB Chadbourne et al. Clinical outcomes in reduction mammoplasty: a systematic review and meta-analysis of published studies. Mayo Clinic Proceedings 2001 76: 503-510.

2 Boice JD et al. Breast cancer following breast reduction surgery in Sweden. Plastic and Reconstructive Surgery 2000 106: 755-762.

3 LA Brinton et al. Breast cancer risk in relation to amount of tissue removed during breast reduction operations in Sweden. Cancer 2001 91: 478-483.

Figure 2: Breast cancer risk after reduction surgery



BIAS IN CRITICAL APPRAISAL - THE EXAMPLE OF ACUPUNCTURE FOR STROKE REHABILITATION

Bandolier 80 contained a short summary of some of the ways that bias can creep into studies, and distort the results. With little exception, the bias is all one way, in that it makes the results look better than they are.

The knowledge that bias can be operating on individual studies, or on systematic reviews if they do not take potential bias into account, means that each of us, individually, has to have our own bias detectors operating continually. In meta-analysis, where data are pooled, sensitivity analysis often tests whether trials with different populations, or trials with different characteristics have different results. In systematic reviews where data are not pooled, an impression about whether a technology "works" is often derived from vote-counting - the number of papers that says it works is bigger than the number that says it doesn't.

Acupuncture for stroke

Suppose we have a proposal to purchase acupuncture for patients with stroke, because it apparently improves their rehabilitation. Being evidence-based, we ask whether there are any randomised trials showing efficacy. The answer we receive is that there are seven trials found from searching databases, and that six of the seven trials show acupuncture works in stroke. It's a no-brainer, we should purchase it.

Hang on a moment

We ask one of our colleagues who has been through critical appraisal training to read the papers. They tell a different story:

- None was double blinded.
- Reporting quality for the six "positive" trials was 2 or less out of 5.
- Validity was 8 or less out of 16 for five of the positive trials.
- Four of the positive studies were done in the far East.

Out colleague tells us that this means that all the positive studies were subject to potential bias. Moreover, when they read the papers, they failed to agree with the original authors for four of the "positive" trials: these trials were actually negative.

So how many trials do we have that were free from potential bias and were positive? Answer is none. Not only that, but our colleague tells us that they are not really sure that it was acupuncture that was being tested. All the trials used "elecro-acupuncture". Three studies mentioned that they tuned up the voltage sufficiently to get muscle twitching, though the other studies probably did it the same way. Is this acupuncture or electrical stimulation of muscles to maintain tone?

Comment

This is a rapid appreciation of a topic that took some time for experienced people to get their heads around. To help readers, the full review is published on the ***Bandolier*** Internet site in html and downloadable PDF formats. When it comes to bias, it's worth remembering that there is a lot of it about.

References:
1 LA Smith, OA Moore, HJ McQuay, RA Moore. Assessing the evidence of effectiveness of acupuncture for stroke rehabilitation: stepped assessment of likelihood of bias. Bandolier publication at www.jr2.ox.ac.uk/bandolier/booth/alternat/ACstroke.html.

Table 1: Acupuncture for stroke: vote-counting with and without allowance for bias

	Conclusion of original authors		Conclusion of reviewers	
Potential source of bias	Positive	Negative	Positive	Negative
No source of bias considered	**6**	**1**	**2**	**5**
Double blind trials	0	0	0	0
Observer blind trials	2	1	0	3
Non blind trials	4	0	2	2
Reporting quality 3 or more	0	1	0	1
Reporting quality 2 or less	6	0	2	4
Validity score 9 or more	1	1	0	2
Validity score 8 or less	5	0	2	3
European studies	2	1	1	2
Far east studies	4	0	1	3

Reporting quality using 0-5 scale [Jadad et al, 1996]; Validity scoring using 0-16 scale [Smith et al, 2000]; Geographical definitions [Vickers et al; 1998]